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DISEASES OF THE NERVOUS SYSTEM

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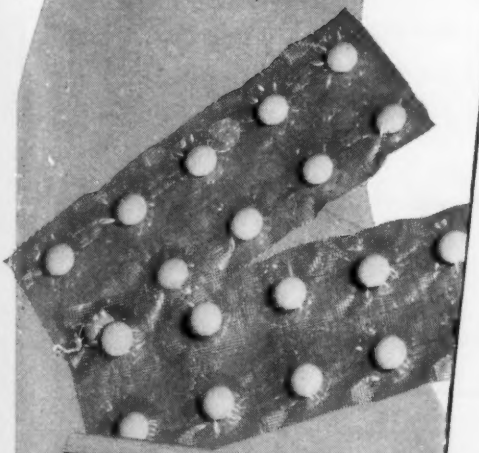

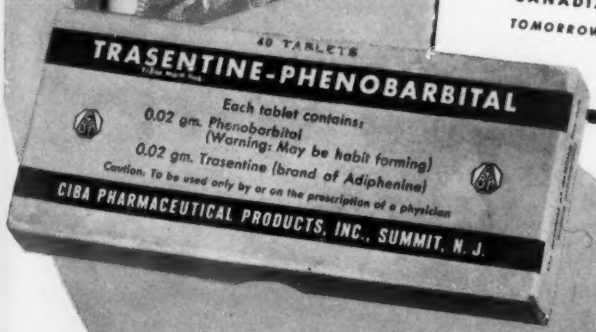
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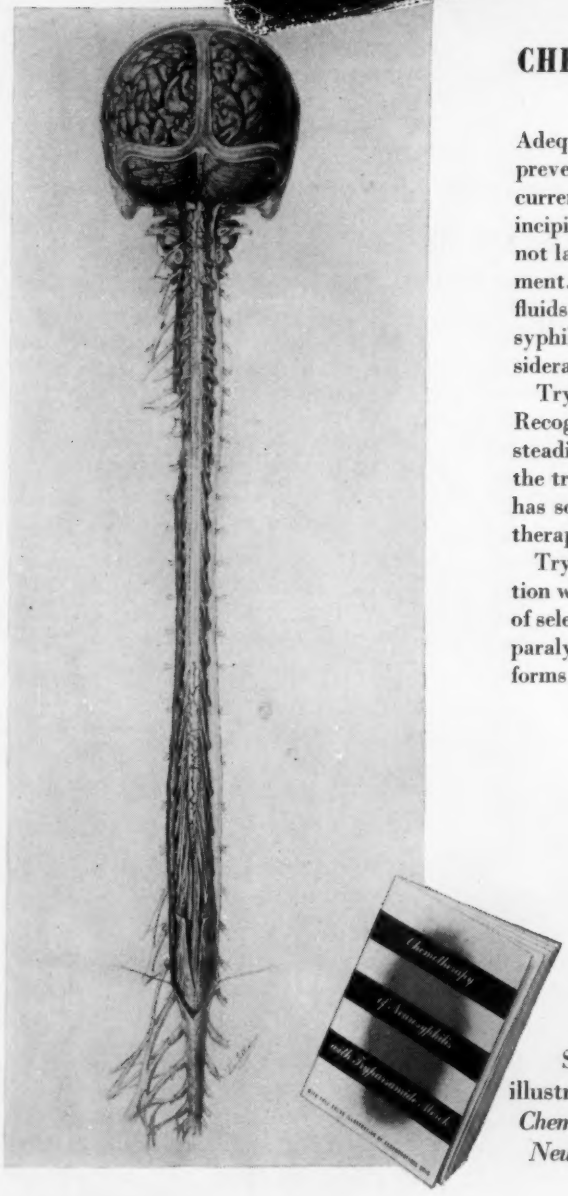

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DISEASES of the NERVOUS SYSTEM

(PUBLISHED MONTHLY)

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

The Centennial Meeting of the American Psychiatric Association

The oldest national medical society, the American Psychiatric Association, has closed its one hundredth annual meeting. The Association met in Philadelphia, the place of its birth, commencing Monday, May 15, and reaching its high point and hour of closing on Thursday, May 18. It was the largest meeting in point of attendance since the founding of the Association, over two thousand people being registered.

The friendly, serene personality of Dr. Edward A. Strecker, the Philadelphian to whom fell the honor of presiding over this historic meeting, pervaded the proceedings. It was a genial, friendly, and festive meeting. It was also a solemn and earnest one. The shadow and the rumbling of a world war were seen and heard at every session, but the spirit of hope and self-reliance and sense of duty was in evidence in every meeting room and in the papers presented.

The war, and the role of psychiatry in preventing and healing our war casualties, were the keynotes of the deliberations. These were led by Major General Norman T. Kirk, Surgeon General of the United States Army, Vice Admiral Ross T. McIntire, Surgeon General of the United States Navy, Major General George B. Chisholm, Director General of the Medical Services of the Canadian Army, our own Colonel William C. Menninger, Director of the Division of Neuropsychiatry, and Commander Francis J. Braceland of the Navy. The immense task imposed by the war upon the American Psychiatric Association and psychiatry as a specialty was discussed in a spirit of freedom, enthusiasm, and a sense of responsibility of which American medicine can be justly proud. President Strecker, serving as consultant to both Surgeon Generals of our Armed Forces, played no minor role in the deliberations.

Despite this overwhelming preoccupation with military problems, the historic and cultural meanings of this meeting were not overlooked. The American Psychiatric Association started in 1844 with a membership of thirteen. It has expanded to a membership of over three thousand. It started as a group of superintendents of mental hospitals; it now is an organization of specialists whose qualifications are well defined and established by the American Board of Psychiatry and Neurology.

A special volume, presenting a comprehensive history of American psychiatry during the past century, was published on this occasion by the Columbia University Press. It is a handsome example of the American art of printing, and was designed by a foremost book designer, Melvin Loos. Doctors J. K. Hall of Richmond, the writer, and Henry Alden Bunker of New York are the General, Associate, and Assistant Editors respectively of this volume. It is good that the history of American psychiatry is put on sale to the profession and to the public in general, since it presents both a history and a living monument to one of the major American cultural values. These cultural values are frequently stifled in the smoke and blood of war. The American Psychiatric Association is to be congratulated on this job of salvaging and preserving some of our national cultural values.

Visions of the great personalities of the Association's past filled the banquet hall and the section meetings. In a special play written for the occasion and acted by a professional cast, the presence of Isaac Ray, the founder of American forensic psychiatry, and Dorothea Lynde Dix, the great spirit of American psychiatry, graced with inspiration and enthusiasm the banquet hall and the job done by a century of untiring effort.

Dr. Alan Gregg, Director of the Division of Medicine of the Rockefeller Foundation, spoke on the Critique of Psychiatry. His was the friendly voice of an honorary member of the Association who summarized for his confreres that censure the membership for the most part has been directing against itself in a spirit of healthy self-criticism and hopeful appeal for further improvement of our work, deepening of our knowledge, and refinement of the awareness of our medical, social, and historical responsibilities.

There was no spirit of self-righteousness or self-laudatory complacency in the meeting. It was a meeting of hard work. It was a period of earnest contemplation of a task to be performed and of the great duties to be discharged in the future, which history has made so near to us by the world crisis.

Gregory Zilboorg, M.D.
New York, New York

Diagnostic Approach to Head Injury Cases

WILLIAM C. PORTER, COLONEL, M.C., UNITED STATES ARMY
JOSEPH FETTERMAN, MAJOR, M.C., ARMY OF THE UNITED STATES

The effect of a head injury may be primarily somatic or psychic, "commotional" or "emotional." Yet secondarily it is a mixture of the two, for as Denny-Brown[1] remarked, "A peculiar mixture of trauma and emotional shock that constitutes all war injuries retains traces of both features throughout the convalescent period." In every injury the mind of the injured man is concerned (Fetterman[2]). With full cognizance of this correlation, nevertheless the distinction as to the basic defect is of practical value. Such distinction will not alone contribute an accurate diagnosis but help towards a program of treatment, decide the period of hospitalization, determine evacuation or return to duty in the combat area.

There is much controversy as to terminology. Such terms as post-traumatic constitution, post-traumatic encephalopathy, post-traumatic general cerebral syndrome, have been applied to the organic syndrome. Each name correctly features one aspect such as constitution, pathologic tissue changes, general symptomatology, yet not quite accurately encompasses the entire picture. Hence for the present we may retain the traditional name of post-concussion syndrome for the organic as a contrast to post-traumatic neurosis for the functional.

Concussion

When a soldier is thrown from a jeep and lands on his head, several pathological processes take place. The sudden arrest of motion by the impact (deceleration) disturbs the position of the brain within the skull. Not alone is there a jar transmitted differentially through the various tissues of the brain, but the brain itself moves in relation to the skull (Denny-Brown and Russell[3]). The deceleration causes a pulling away from one side of the cranium, a striking against the opposite wall and possible twisting of the brain stem (Rowbotham[4]). The sudden loss of consciousness, according to Symonds,[5] is due to a disturbance in neurone function produced by stretching and compression. Guitierrez-Mahoney[6] has confirmed the experimental observation that the abnormal movement of the brain within the skull resulting from sudden acceleration or deceleration is responsible for the more serious damage. A blow to the head of an animal held fixed causes fewer lesions than when the head is accelerated or decelerated.

A sudden blow which strikes the head and jars its contents produces not only a paralysis of neurone function but also generalized edema. Many factors contribute to the edema which involves ganglion cells, the pericellular spaces and the perivascular spaces. According to Hassin[7] and Courville,[8] and others, the edema results from vasomotor instability, altered osmosis between the cells and the surrounding spaces, and altered production, circulation and absorption of the cerebrospinal fluid. In mild trauma such changes are reversible. When the injury has been more severe there may be petechial hemorrhages and more gross contusions of the surface of the brain.

The immediate consequence of a closed (non-penetrating) head injury is usually unconsciousness. This is followed by a series of symptoms which depend largely on the severity of the injury (and, as always, upon the personality make-up of the individual). The patient awakens from the unconscious state dazed and bewildered; he looks about and remembers that he had just started on a journey or that he had just gone to bed and now he finds himself in the hospital. At first he does not know why or how he got there, then as his awareness increases and his orientation improves, he becomes conscious of a severe throbbing headache and vertigo.

Within a period of days his mind becomes alert and his distress tends to decrease. However, for some days symptoms of fluctuating intensity persist. The headache and dizziness are made worse when he moves suddenly; strong lights are blinding, and sounds annoying; in other words, his threshold of awareness is low. Concentration is difficult and he fatigues readily. Effort, either physical or mental, makes him uncomfortable. Exercise and drinking add to his distress. In the course of time the symptoms tend gradually to abate.

They return, however, during episodes of excitement, challenging tasks and difficult problems, and he then becomes upset, irritable and uncomfortable. These episodes decrease in frequency, and there is a gradual return to normalcy. (In those instances of more severe injury involving laceration and intracranial hemorrhage, gliosis and cyst formation may result in defect and permanent residual symptoms.) Then we encounter loss of memory, fatigability, diminished attention and concentration and focal symptoms. The disorder may range from a minor defect to a profound change in personality and in the capacity to plan and execute.

Neuroses

The patient who was frightened by a shower of sand from a distant bomb blast may complain of similar symptoms. However, such complaints may be merely the expression of a personality unable to cope with his environment.

The quality and pattern of a psychoneurotic personality are usually developed early in life. A succession of factors has contributed to this personality make-up. Constitutional tendencies determined the quality and the dynamic energy. For inheritance not only shapes our features but also influences physiological and emotional reactions. Childhood environment exerts molding pressure. A home atmosphere of insecurity, the uncertainty between periods of parental affections and outbursts of anger, parental examples of neurotic complaining condition the growing child. Ultimately it is the child's method of meeting problems that determines his personality. From infancy onward a child grows up in an environment and a setting which provides both love and hostility, where instincts are met with denial as well as with gratification. Some resolve this conflict readily, but others fail to make adequate adjustment and can restrain aggressive impulses only through fear of punishment which leads to the use of symptoms as a compromise. As aptly stated by Kaufman,[9] "neuroses are linked with emotional patterns evolved early in a child's adaptation between instinctual drives and environmental forces. . . . Frustration evokes aggression and the fear of punishment requires a repression of such aggressive instincts." According to this author, neurotic symptoms represent a compromise which includes a partial gratification and yet punishes.

The sum total of inheritance, training and the individual's own early reaction patterns shape the psychoneurotic personality and determines his "constitution." An outstanding feature is a pronounced tendency toward fear reaction resulting from an inner sense of guilt or from outward reactions. The normal pleasure reaction is restricted because of the fears that are inherent in every exposure of participation. There is an imperfect growth of character with a failure of complete development of sex drives and adult responsibility. There are tendencies to revert to an infantile dependent attitude. Such a person is likely to be concerned about the function of his organs and also about various diseases. He is preoccupied with himself and craves attention. Fears and worries replace normal self-reliance. There is a tendency to fall sick with symptoms of a nervous type when faced with conflict. A psychoneurotic individual need not be consistently ill, however, for he may get through periods with little or no complaining. His symptoms may be held in check by ambition, by precautions, or by treatment. But under stress and strain and exhaustion, when external danger mobilizes inner fear reactions, when the discomforts of actual disease or the pain of injury occur, the tendency to develop symptoms asserts itself. On such occasions the potential neurotic becomes a sick man. He tends to utilize illness or injury to solve inner problems. The terrors and hazards of combat are a culture medium for the growth of neurotic symptoms.

The occurrence of trauma affords a chance for conversion of the potential into the real. Miller[10] emphasized the vital influences of the early patterns of fear and insecurity and the need for affection and protection as the determinants of neurotic symptoms. According to Schilder,[11] a blow to the head may evoke concern about sanity or may furnish a nucleus around which neurotic attitudes crystallize. Thus the neurotic who has received a blow on the head will become preoccupied by the pain, the swelling and the fears of possible consequences. He will complain of tightness and pressure, he is troubled by dizziness and inability to remember and think. He becomes weak, sleepless, and worries and frets about losing his mind. He craves sym-

pathy and demands attention through verbal statements or by the more dramatic appeal of hysterical manifestations.

Essential Differences

Though there is a similarity in symptoms, the primary basis is seen to be different. The neuroses are essentially psychogenic, the post-traumatic concussion physical. This difference makes the distinction almost self-evident in many instances. The patient with the organic syndrome suffered a fairly severe injury with evidence of brain damage; the neurotic had the constitutional tendency to illness and his injury may have been only superficial.

In actual practice, this difference is not so self-evident. Following a blast a soldier lost consciousness and bruised himself in the fall. Later he seemed to be in a fog. Did he faint from fright or fall unconscious from intracranial edema? There were no observers available to determine the probable physical violence endured or to record the symptoms. We must rely upon our own analysis to formulate an opinion.

As we seek for differences we must not lose sight of combinations of organic and functional. The two may have been concurrent or in succession. A neurosis may have been superimposed upon an organic head syndrome in the same way as a hysterical paralysis may develop after a fracture of the fibula. In the latter instance we may attempt to separate the invalidism from the basic disability which arises from the fracture proper (say six weeks) from that contributed by the psyche, which may continue indefinitely. So, too, in the head injury cases, we must attempt to distinguish the functional from the organic, however difficult the task.

Methods in the Different Diagnoses

In some instances the distinction is simple and can be made by considering a few positive signs. In others the problem is involved and one must utilize a veritable battery of procedures from an intensive history to a thorough examination and many laboratory tests. A concrete differentiation between organic and functional was outlined by Kennedy[12] some years ago. Kennedy listed a series of findings which were indicative of organic disease. His absolute criteria included roentgen evidence, bloody spinal fluid, and bleeding from the orifices. The presumptive criteria were convulsive states or ventricular distortion proved to be post traumatic and a history of prolonged unconsciousness and adequate trauma. We should like to extend these criteria and to emphasize the importance of obtaining evidence of neurosis by positive data, not merely by exclusion. Let us consider several items:

1. The Pre-Traumatic Personality.

It is uncommon for neuroses to make their debut in adult life following stress and strain, an illness or injury. More frequently the individual has, in his earlier days, revealed traits and habits of illness which stamp him as neurotic. Gillespie[13] has outlined a series of traits of childhood which are the fairly common precursors of adult neuroses. Gillespie's list includes pronounced morbid fears, timidity and lack of aggressiveness, habitual anxiety, inferiority feelings, and physiological instability in the form of habitual restless sleep, stammering, nail biting, bed wetting, fainting at the sight of blood, and excessive visceral reaction to emotion.

The above list of traits does not apply to the early life of every neurotic, nor is the normal person entirely free of them. However, the history of a majority of such characteristics and of previous illnesses suggestive of neurosis or hysteria is weighty evidence for functional illness. The examiner should make every effort to obtain a complete history. Though it seems time-consuming, such a history will actually abbreviate the examination and accelerate the recovery. Symonds[14] makes the point that the discovery of psychopathic traits in the patient's personality will tell much more than an air encephalogram or other special procedures. "What's past is prologue," reads an inscription on the Archives Building in Washington. This is equally true for individuals as for nations.

2. The Type and Severity of the Trauma.

Although a minor accident might lead to serious consequences (subdural hematoma), yet, all things being equal, the severity of the injury will usually determine the seriousness of the symptoms. A soldier hurled from a jeep and striking his head against a rock will suffer far more seriously than a man in the quartermaster corps whose head is bumped by a carton. Yet recently a soldier who sus-

tained a minor bruise complained much more than the patient in the adjoining bed whose injury was a laceration of the brain.

3. Alertness versus Amnesia at the Time of the Accident.

Loss of memory for the experience prior to the injury (retrograde amnesia) is the rule in a concussion of any severity. Indeed a patient may not be able to recall his activities for several hours, or even days, before the trauma. In neuroses there may be no amnesia. On the contrary, the neurotic patient may describe vividly how frightened he was, how he worried that the careless driver of the car would get him into danger (Fetterman[2]). His worrisome awareness of all the details surrounding the episode may be remarkable. Pre-traumatic alertness and apprehension suggest a neurosis; pre-traumatic amnesia points emphatically to organic pathology. A striking exception is the retention of memory and consciousness in some penetrating injuries of the skull by a bayonet and the shearing of part of the skull and brain by a swiftly moving propeller blade (Rowbotham). Goldstein[15] mentions instances in which a bullet of swift velocity penetrated the skull without causing loss of consciousness.

4. Unconsciousness and Delirium.

Loss of consciousness characterizes concussion. The disturbance in ganglion cell function from edema and vascular change consequent upon stretching and compressing of the brain blots out consciousness. Indeed the duration of this loss of consciousness is a useful index of the severity of the injury. (Pilcher and Angelucci[6]). Unconsciousness lasting over three hours is considered by Courville as an indication of hemorrhage within the brain.

After a severe injury the patient goes through a clouded state and a stage of confusion and delirium before he becomes fully conscious. Memory for this phase is usually blotted out. Cairns[17,18] uses the duration of this phase of post-traumatic amnesia as a prognostic time-table. According to Cairns, if the unconsciousness lasted five minutes to one hour, recovery can be expected in some six weeks; if the amnesia extends several days, the recovery period will last up to four months. When the amnesia extends over seven days then the prognosis is poorer and improvement may take four to eight months. It is obvious that loss of consciousness reflects organic change, whereas a momentary dazing or no loss of consciousness is the rule in the neurotic reaction.

5. Neurologic Signs, Physical Findings and Laboratory Data.

If a record of the initial examination at the time of the injury is available, it may furnish positive evidence. We will learn not only about unconsciousness and delirium, but about bleeding from the ears and nose, the presence of unequal pupils, twitchings and paresis, and the findings in the spinal fluid. Later we may obtain significant information from skull films, residual neurologic abnormalities and other tests. Sometimes a pneumoencephalogram will reveal the positive evidence of organic damage, but its use must be limited to definite indications (suspicion of subdural hematoma, epilepsy, and severe headache). Such evidence, of course, reflects more serious cerebral damage than has occurred in the usual post-concussion state and represents a contusion or laceration of the brain.

The study of the brain waves by electroencephalography promises to be remarkably helpful in ascertaining the site and severity of brain damage by trauma. Williams[19] has demonstrated by serial brain wave studies the changes which are revealed in the early post-traumatic period and the improvement in the rhythm of such waves which runs parallel to recovery. Ultimately this procedure may simplify our problems, affording pictures of comparative value.

Other procedures may be utilized to establish a diagnosis. Goldstein's recent book outlines several tests which reveal defects of attention and memory. The Rorschach Ink Blot test may also be used because there are responses considered characteristic of organic damage. (Piotrowsky;[20] Bochner and Halpern[21]). Psychological tests which reveal loss of previous capacities can be employed to reveal impairment in brain function.

6. Special Symptoms.

Following a concussion the symptoms of headaches, dizziness, weakness, and vasomotor instability have certain features. For example, they are made worse by movement, by heat and by drinking. On the other hand, a psychoneurotic may say that his head bothers him more when sitting idly, exercise in the

warm sunshine makes him feel good and a few drinks make him happy. Several of the most severe head injury cases had no headache but marked loss of interest, undue cheerfulness, profound memory loss. Loud complaints of headache were common in the neuroses.

Other symptoms of the psychoneuroses include conversion disabilities, for example, paralysis and loss of sensation, altogether out of line with the organic pathology. The psychoneurotic may complain of loss of vision or loss of sensation in the entire half of his body where he was struck (conversion hysteria). His hearing, vision, and gait may be peculiar. The specific and the general symptoms do not conform to a characteristic organic syndrome.

7. The Mental Attitude and Thought Content in the Later Period.

The average person who has sustained head trauma is obviously distressed by his symptoms, yet he seeks help hopefully—he makes the best of his condition, tries to do as much as he can, even in a restricted manner. He looks forward toward hope of recovery, he directs his energy toward effort. Not so, the psychoneurotic patient. Attention is concentrated upon the area injured. There is abnormal concern about sanity, paralysis or other dire consequences. The patient's major interest is now his head; the future looks dark; he shows undue dependence and attachment. The injury occupies the foreground of consciousness and reactions to the injury reappear frequently in dreams. (Kardiner[22]). Terror dreams featuring violence recur.

The preoccupation with the injury, the pessimistic mood, the tendency to blame others—these attitudes are more or less pronounced in psychoneuroses. The normal individual who has sustained concussion will also be concerned about it and will show some of the above reactions. The intensity, persistence and preoccupation characterize the psychoneurotic.

8. Gain: Advantage of the Symptoms to the Individual.

Injury concerns the individual in his relation to his environment. The disability prevents the normal response to work and rest, disrupts plans and purposes, directs attention from outside problems to inner pathology. In civilian life, someone may be responsible for the injury and there is an instinctive effort to obtain redress. A claim for compensation may become a motivating force in the life of the injured person. During war time an injured soldier is taken from the dangers of the combat area, is withdrawn from the usual pressure of training, marching and fighting. He is placed in a relatively safe environment and receives sympathy, warmth and attention. Whether the gain be monetary in civilian life or safety during war time, it is a factor which tends to prolong the symptoms. In the history of psychoneurotic illness, the element of gain looms large. The tendency to place the blame upon others, and the tenacious clinging to symptoms may serve an inner need. However, there is often a material advantage to the illness. The item of gain is an incentive in another group of cases. We refer to the sociopathic (the constitutional psychopathic) individual. Such a person uses the injury as an excuse for the further practice of his philosophy of life, namely, that society owes him a livelihood, and that any reason for his failure or nondesire to fulfill his obligations to society is sufficient and should be utilized to the greatest possible extent. This is an important consideration in those accident cases where the compensation may become an issue. There is a mixture of conscious simulation and wishful thinking. This mechanism should be differentiated from that present in the psychoneurotic individual where motives are largely unconscious.

9. Sequence of Events:

As a rule symptoms of a concussion are more intense at first and in the course of time they lessen. In the moderate case there is likely to be considerable relief and tendency toward further recovery. Cairns[17] speaks of the common cycle which begins with unconsciousness, passes through a stage of confusion, a period of headaches and finally reaches the point of more or less total recovery. Such a curve of recovery is not smooth and steadily upward, but there are fluctations from time to time. These variations brought on by inner factors or external events, do not alter the trend of progress from ill health to well being. Not so in neuroses. The symptoms are markedly influenced by outside forces. They are responsive to situations. For the average person, time is a therapist;

in psychoneurosis it may be an opportunity for growth of symptoms.

Diagnostic Steps Under Combat Conditions

The combat situation surrounds the soldier with the hardships which induce neurotic reactions even in the normal. The trials of heat and cold, exhaustion and hunger, noise and filth, bombs from overhead and bullets from unseen enemies creeping stealthily from the jungle, danger and death lurk everywhere. Such a situation obviously causes visceral and vasomotor reactions which may be the starting point for a neurosis. Likewise, bomb blasts may cause injury as do bullets themselves. The diagnostic problem from blast concussion is quite a challenge, especially so when we picture the flow of wounded during combat and the limited medical conditions which prevail under such circumstances. The surgeons at the zone of combat must rely on several practical points: A brief history furnishing a clue to the personality, an inquiry as to the mechanism of the injury. Was the soldier struck by a bullet; was he hurled to the ground; was the bomb crash near or far distant? Lt. Colonel Berezin[23] described certain neurotic soldiers who became startled by the sound of bombs at a distance as well as by explosions nearby. One should learn, if possible, if there were unconsciousness, how long did it last and what were the stages in the return to consciousness. Next comes a study of the immediate symptoms, and a thorough scrutiny of the patient: any signs of trauma, bruises and bleeding, neck rigidity, color, pupils, facial asymmetry, twitching, differences in tendon reflexes, abnormal reflexes. Any positive findings should be noted and recorded.

Let us consider the case history of a soldier who was evacuated from Guadalcanal to the Zone of the Interior. In the fox holes of Guadalcanal he had been unhappy, trembly and fearful. Heat, disturbed sleep, monotonous rations added to his distress; the thought of Jap snipers frightened him; he became jumpy when leaves rustled as well as when bullets were fired. Then there was an explosion and he was jarred backward. He believes "he was dazed but regained consciousness" and walked back to the battalion aid station and complained of sickness. He developed tremulousness and headache. These symptoms grew steadily worse. Even after evacuation to the United States his symptoms became worse and worse.

When examined in the U. S. his complaints included headache, sleeplessness, weakness, inability to concentrate, fear of noises. An exhaustive examination proved negative. Then we chanced to see him on the day he had received his discharge. For the first time a smile replaced the dull cloud on his countenance. The earlier opinion at the combat area was supported by the more extensive procedures at the General Hospital to the effect that the symptoms represented a neurosis.

Diagnostic Methods at a General Hospital

Here are available facilities and personnel for a searching study. As a supplement to the routine clinical features one may utilize the talents of specialists and a battery of laboratory tests. Here can be employed detailed x-rays and lumbar punctures, pneumoencephalograms to reveal gross defects in brain structures or an electroencephalogram to show alteration in function. Equally significant are tests of retention and recall, of judgment and insight, of emotion and drive. Though life itself is the crucial test of an impaired brain, yet these procedures tell us much about the intactness or injury of the brain. For example a corporal was a passenger in a jeep which ran off the road. He was found unconscious and remained thus until he was put to bed in the station hospital. When he recovered consciousness several hours later he developed nausea and vomiting. A transitory weakness was found in the right half of the body. Gradually he improved. Later he was sent to the Lawson General Hospital. He had few complaints except a minor headache. The x-rays and lumbar puncture were negative. However, even two months following the injury there were residual neurologic signs: diminished associated movements in the right half of the body, unequal pupils, a difference in abdominal reflexes; positive Chaddock in the right foot. But of greater meaning was a certain dullness of features, an apathy. Some considered it a neurosis. Yet clearly the positive neurologic signs were supported by a profound defect in memory and by a Rohrschach test of an organic pattern. Thus the psychologist, Lt. Hughes, summarized his findings as follows:

"The patient presented a rigid, constricted and blocked response pattern which is typical of the performance in cases with extensive intracranial organic pathology."

Treatment

Though the subject of treatment will be dealt with in another paper, one may remark in passing that diagnostic procedures are an aid to therapy. A careful examination aids in establishing rapport. Likewise the findings decide the measures to be used and what can be expected of treatment. Therapy has several aims in attaining the ultimate goals of rehabilitation: to assist in restitution towards normal of the injured tissues; to guide the mental attitude towards a will to get well. To accomplish tissue repair we must avoid infection, reduce increased pressure, provide sufficient rest. To foster the will to get well we must avoid ideas of calamity and encourage a feeling of optimism, of effort, of independence. To guide the convalescing person towards usefulness we must influence contacts to assist and encourage. For the soldier we should cultivate a will to return to duty, for the civilian offer incentives to work such as the opportunity for a good job not a pension for prolonged disability.

Conclusions

This paper has discussed the basis for symptoms following concussion and those following trauma in a psychoneurotic personality. The psychoneuroses are psychogenic, the post-concussion state physiogenic. Often the distinction is difficult and can be made only by a consideration of several criteria. A distinction should be made, not on one item alone, but by a careful weighing of all the facts. The criteria mentioned are pre-traumatic personality, the severity of the injury, pre-traumatic alertness or retrograde amnesia, unconsciousness, neurologic and laboratory evidence, the grouping of symptoms and the mental status, the factor of gain and the sequence of events. Careful weighing of all evidence should enable one to arrive at a just evaluation upon which to base a program of treatment.

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Certain Medico-Legal Aspects of Psychiatry*

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Medical students and general practitioners frequently ask: "What about this claim that patients are 'railroaded' into state hospitals?" "Who can commit a patient?" "How is it possible to get a patient released from a mental hospital after he is committed there?" "How can a physician get a patient into a mental hospital when the patient doesn't want to go and when he is threatening to sue anyone sending him there?" "Is it possible to commit alcoholics to state hospitals?" "Does one have to be a specialist in psychiatry to sign commitment papers?" "Is there any difference in the professional qualifications required of a physician testifying in criminal cases as contrasted with non-criminal cases?" "How are the property rights of a committed patient protected?" "Is it possible to divorce a committed mental patient in New York State?"

In this paper I shall try to answer these questions. For an official legal reference to these problems I would refer you to *"Handbook of the Department of Mental Hygiene,"* published by the State Hospital Press, Utica, New York.

In New York City, any physician, public official (policeman, etc.) or the relative of a patient may request the admission of a patient to the Psychiatric Division of Bellevue Hospital or Kings County Hospital for emergency treatment. The patient is examined by the psychiatrist in charge of the admitting ward and this psychiatrist determines whether or not such admission is necessary. If the patient is admitted, he may be held in the hospital for mental observation up to thirty days. Prisoners and juvenile delinquents may be committed to these hospitals by a judge of a court of record for a period of mental observation varying from ten to thirty days. In other parts of New York State any person alleged to be insane to a degree which warrants institutional care and who is not confined on a criminal charge may be admitted to and confined in a state hospital or a state psychopathic hospital or a licensed private institution through one of the following five procedures: (1) voluntary application; (2) certificate of a health officer; (3) certificate of a certified examiner; (4) court commitment; or (5) an incomplete court commitment to meet an emergency. If a physician or two physicians make petition for such emergency admissions, they must be *qualified examiners* in lunacy.

If the individual thus admitted for emergency treatment demands his release, the physician in charge has the right by law to detain him from ten to thirty days, depending on how he was admitted. The patient, his lawyer or relatives, however, may secure a writ of habeas corpus and he is entitled to a hearing in the Supreme Court in the county in which the patient is confined. The Supreme Court Justice may dismiss the writ or he may order the release of the patient.

If the physician in charge believes that the patient is suffering from a transitory mental illness such as a mild reactive depression, alcoholic psychosis, a mild delirium with somatic disease, etc., he may release the patient into his own custody or that of relatives or friends until the patient has recovered from the illness. He may also discharge the patient *against advice* to a relative or physician if he thinks the patient is mentally sick but not potentially dangerous to himself or others. The person to whom such a patient is released signs a notarized agreement wherein it is stated that he realizes he is removing an alleged incompetent person from the hospital and is willing to assume the responsibility for the patient's care.

If the physician in charge of the patient in a psychopathic or state hospital decides after a period of observation that the patient will require prolonged care in a mental hospital he may arrange for the patient's *voluntary admission* or his *commitment*, to a state or private hospital. If the patient is an infant, i.e., under 21 years, his parent, next of kin or legal guardian must make an application in writing for *voluntary admission*. The voluntary application sets forth that the patient is willing to

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enter or remain voluntarily in a mental hospital (state or private) and that he will give ten days' notice in writing of his intention or desire to leave the institution.

If the patient is to be *committed*, the nearest relative or guardian is requested to sign the application for commitment on a special form provided by the Department of Mental Hygiene. The relative makes a statement of his observations which led him to believe that the patient had mental symptoms and requires commitment. If the relative or "next" friend refuses to sign the application, he is served with a notice, personally or by mail, informing him that on the sworn petition (affidavit) of the superintendent or deputy superintendent of the hospital where the patient is confined, and upon the certificate of two *qualified examiners* (whose names are there mentioned) application will be made before a certain specified court on a specified date for the commitment of the patient to a mental hospital. The relative or friend must receive notice of the application at least one day before the hearing.

The patient himself is also served with a similar written notice at least one day before the court procedure so that he or his relatives may make plans to protest the commitment by securing attorneys, outside psychiatrists, etc.

The *commitment papers* contain a statement as to how the patient was admitted to the hospital, a brief history secured from outside sources as well as a detailed statement of his physical and mental condition and verbatim statements of the patient made to the two *qualified examiners* jointly which indicate the presence of delusions, hallucinations or other psychotic symptoms.

Voluntary admission requires the signature of only one qualified *examiner* but the commitment paper must be signed by two *qualified examiners*. A *qualified examiner* is defined by the Mental Hygiene Law as: "a reputable physician, a graduate of an incorporated medical college, duly licensed to practice medicine in this state, who shall have been in the actual practice of his profession for at least three years at the time of his certification as such, . . . qualified examiners . . . shall be authorized to act as such when the facts of their qualifications shall have been certified by a judge of a court of record in a form prescribed by the commissioner. The original certificate shall be filed in the office of the clerk of the county in which the qualified examiners . . . reside, and a certified copy thereof filed and recorded in the office of the department of mental hygiene and its receipt for such filing and recording duly acknowledged." It can thus be seen that a *qualified examiner* need not be a specialist in mental disease.

If neither the patient nor his relatives or friends request a hearing to oppose the commitment, the judge signs the commitment order which is forwarded to the state or private hospital and the authorities from that institution send their transfer agent to conduct the committed patient to the appropriate hospital.

If a hearing is demanded, the patient, with or without relatives, attorneys, or physicians, appears before the judge. One of the qualified examiners testifies as to his findings and gives his opinion as to the need for commitment. The patient and/or his representatives then testify and the judge from the testimony adduced commits or refuses to commit the patient or he may adjourn the hearing to give the patient or his relatives or friends opportunity to secure lawyers, physicians, etc. If the judge refuses to commit the patient, the patient is released unless he is a prisoner (in which case he is returned to the court from which he was originally remanded.

If the patient is committed, he has the right to demand a *review proceeding* within thirty days before a different judge. If the second judge sustains the order of commitment, the patient still has the right to a *trial by jury*. The review proceeding and jury trial may occur even after the transfer of a patient to a state hospital provided it be done within thirty days. If the second judge or the jury finds the person sane, he is ordered released but if he is found insane the justice shall certify that fact and make an order for recommitment.

When the person is finally committed and becomes an inmate of a state or private mental hospital he may be released only if the superintendent of the institution believes the patient is recovered or improved to the extent of no longer being a potential menace to himself or others. When a committed person is so released, he is usually placed on parole for one year; he is required to report for check-up at

periodic intervals and must have a final examination before he is finally released from parole. He may, however, be released outright without parole. If there is a relapse during the parole period, he may be returned to the hospital without court proceedings. At any time while the committed patient is in a mental hospital he or his relatives have the right to secure a writ of habeas corpus and have a hearing in a court of record to establish the mental condition of the patient.

These procedures for the commitment of the insane apply also for the commitment of defectives and of epileptics, except that these latter are committed to state schools or epileptic colonies unless they are psychotic as well as defective or epileptic. Moreover, defectives may be committed upon testimony of one *qualified examiner* and one *qualified psychologist*. (The qualified psychologist must have two full years of postgraduate study in psychology at an incorporated university or college and three years of actual clinical experience at the time of his certification.)

Occasionally the court will commit a patient to an individual or group of individuals, technically known as *"committee of the person."* Such individuals have the same duties as *"committee of the property"* which I shall describe later.

What about the commitment of alcoholics? If these individuals have a chronic psychosis, such as deterioration, a Korsakoff syndrome or a chronic hallucinosis, they may be committed to state institutions. If they are merely alcoholics, they may be committed to private hospitals in New York State but may not be committed to state hospitals. Occasionally chronic alcoholics who are suicidal or homicidal when intoxicated are committed with label of "Psychosis with Psychopathic Personality" to state hospitals but such individuals are usually released outright from state hospitals within a few weeks.

In *criminal cases* where patients are committed to psychopathic or state hospitals for mental observation a different procedure is followed if such individuals are found to be mentally sick. In these cases the law requires a distinction between a person who is *mentally sick* (psychotic) and *legally insane*. According to the Code of Criminal Procedure, a person is considered to be insane when he is so disturbed mentally that he doesn't know the nature and quality of his acts and is not able to advise with an attorney in his own defense. According to the strictest interpretation of this law only a markedly deteriorated schizophrenic or a patient with an organic psychosis who was confused, forgetful, etc., could be considered "insane." However, most psychiatrists place broader interpretation on this law and consider that a person may be so motivated by his delusions and hallucinations as to be incapable of understanding the true nature of his acts. It is precisely because of the conflicting interpretation of what constitutes legal insanity that so much newspaper publicity is given in the so-called "Battle of Experts" in murder trials, etc.

In New York State the Code of Criminal Procedure permits only *qualified psychiatrists* to make official psychiatric examination of prisoners. These *qualified psychiatrists* are appointed by the director or superintendent of the psychopathic hospital or state hospital where the prisoner has been sent on court order for a mental examination.

To be a *qualified psychiatrist* in New York State one must be a physician duly licensed to practice in the State of New York and one who has had at least five years' experience in actual practice. In addition, he must have had: (1) two years of full time practice since January 1, 1920, in the care and treatment of persons suffering from mental diseases or mental defects in an institution providing for the care of such persons and having accommodations for at least fifty such patients, or (2) he must have devoted the five years immediately prior to filing his application for certification to a practice confined wholly or substantially to the care and treatment of persons suffering from nervous and mental diseases or mental defects, or (3) he must have had three years' experience in a clinic approved by the board devoted to the diagnosis and care of mental disorders and his competency must have been certified by two psychiatrists already certified by the Board of Psychiatric Examiners.

If prisoners examined by these qualified psychiatrists are found to be insane within the meaning of the law, special procedures must be followed out. The two qualified psychiatrists either report their findings in writing to the court or arrange for the commitment of the prisoners directly to mental institu-

tions and then notify the court after the commitment has been made. The procedures and eventual disposition of the prisoners vary with the nature of the criminal act and the jurisdiction of the criminal court before which the prisoner is being arraigned, his previous criminal record, and his clinical nature. If very disturbed, he may require treatment in a hospital for the criminally insane rather than in a civil state hospital. (Code of Criminal Procedure, Sections 658 to 870, as amended by the New York State Legislature in 1939.)

How are the property rights of mental patients protected? After a patient is committed his nearest relative or friend may petition the court for the appointment of a *committee of the property*. This committee consists of one or several persons appointed by the court on proper application. They must file in the office of the clerk of court, in which such a committee was appointed, a bond with at least two personal sureties or a surety company in an amount fixed and approved by the court. The bond provides that the committee "will faithfully discharge the trust imposed upon him or them, obey all directions of the court in regard to the trust, make and render an account of all moneys and properties received, etc." The committee of the property cannot mortgage or dispose of real property, except to lease it for a term not to exceed five years, without the specific direction of the court obtained upon proceedings taken for that purpose. The committee may be suspended, removed or allowed to resign at the discretion of the court.

When the patient becomes competent to manage himself or his affairs, the court will make an order discharging the committee of person or property or both and such committee must render and file an accounting of his management of the incompetent's property.

What about *divorce* or *annulment proceedings* when one of the parties in marriage is mentally sick?

An *annulment* of marriage may be possible in New York State if it can be proved that one person was psychotic (the law uses the term "lunatic") at the time of the marriage and the partner was not aware of the condition. The annulment may be secured at any time after the marriage unless "the parties freely cohabited as husband and wife after the lunatic was restored to a sound mind."

A marriage may be *voided* if it can be shown by competent medical opinion that one party in the marriage has been "incurably insane" for a period of five years or more. However, if the wife is insane, the court, after rendering judgment, must exact security to be approved by a judge of the court, for her suitable care and maintenance during life.

Before a marriage can be either annuled or voided besides other proof of mental disease there must be a thorough examination of the alleged insane party "by three physicians who are recognized authorities on mental disease, to be appointed by the court, all of whom shall agree that such a party is incurably insane and so report to the court." If the mental patient is in a state hospital, one of the three physicians must be a member of the resident medical staff of the hospital. If the patient is not in a state hospital, one of the three examining physicians must be a superintendent of a state hospital."

Conclusion

In this article describing procedures dealing with the admission and commitment of patients to psychopathic or state hospitals or private sanatoria, I think that it is clearly demonstrated that the laws of the State of New York provide ample protection for mental cases and that it is very difficult to "railroad" a mentally competent patient into a mental hospital.

Editor's Note: Dr. Curran writes with intimate knowledge of the legal methods and requirements in New York State. He has first hand acquaintance with the material he presents by virtue of his position and associations. Comments on the above or on methods or procedures in other states, where, perhaps, the laws are not as complete or comprehensive will be welcomed.

Vasomotor Neuroses*

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A number of poorly defined syndromes presumed to be a combination of peripheral nerve, blood vessel, and sympathetic nerve disorders have been included under the title of vasomotor neuroses. Such diseases as acroparesthesia, Raynaud's disease, angioneurotic edema, erythromelalgia, scleroderma, causalgias, phantom limb, and other indefinite syndromes have been so classified.

The etiology of all these conditions is obscure. The autonomic nervous system with its effect upon peripheral blood vessels and consequent production of vasoconstriction in some cases, vasodilation in others, and certain types of pain, seems to be a causative factor. Whether the endocrine system and other metabolic disturbances play an etiologic role is undetermined. A neuropathic predisposition with an hereditary diathesis may possibly explain in part some cases. Exciting factors such as trauma, focal infection, and exogenous toxins probably start the vicious cycle of abnormal neurovascular physiology.

To a great American neurologic pioneer, S. Weir Mitchell, should be given credit for accurately describing most of these syndromes; some bear his name. During the Civil War Mitchell, Morehouse, and Keen studied patients with peripheral nerve lesions and described such conditions as causalgia, phantom limb, reflex paralysis, and erythromelalgia. Later they established the Orthopedic Hospital and Nervous Infirmary of Philadelphia, where I had the privilege of serving my neurologic residency. To Weir Mitchell's descriptions seventy years ago of these disorders we have added but little in our clinical observations or laboratory research toward a better understanding of these obscure disorders.

Acroparesthesia

This disorder is usually seen as part of a menopausal syndrome in women. The symptoms, largely subjective, consist of numbness, coldness, tingling, and crawling sensations of the extremities, especially the fingers. At times the sensations are painful; the hands feel stiff and are usually worse at night. There are no objective signs except slight pallor. This is a vasomotor neurosis sometimes resembling tetany. Apparently an irritability of the vasomotor centers results in constriction of the blood vessels and produces slight anemia of the peripheral nerves.

The disorder runs a chronic course and must be differentiated from such organic diseases as spinal gliosis, combined sclerosis, and acromegaly. At times it is associated with polycythemia. Often seen with emotional instability, it is sometimes hard to differentiate from an hysterical neurosis.

Raynaud's Disease

This disease is sometimes called symmetrical gangrene. In the early stages a dead feeling occurs in the extremities with attacks of local syncope or asphyxia. This condition is followed by cyanosis and later by a blue black appearance of the fingers or toes with a sharp line of demarcation. The majority of cases occur in young women. The cause is unknown. At times the disease is seen as a symptom of organic neurologic diseases such as multiple sclerosis, syringomyelia, cord tumor, or tabes dorsalis. The vasomotor disturbance may be secondary to some endocrine disease. At times attacks of frostbites precede the syndrome. Cold weather or occupational factors make the symptoms worse.

The attack generally begins with numbness or deadness in the fingers or other symmetrically situated parts such as toes or lobes of the ears. At first the parts are pale and waxy; they feel cold, with local syncope. There may be intense pain during this period. The attacks last from minutes to hours and are relieved by hot applications. The syncope is replaced by cyanosis, the skin becomes bluish red, and the pain increases. Other trophic changes of skin and nails with atrophy of muscles may occur and dry gangrene and ulcerations eventually result. Insomnia, anorexia, loss of weight, and emotional upsets are disturbing general symptoms at times. The attacks recur over a period of years before gangrene finally develops.

*Presented before the Omaha Midwest Clinical Society.

Erythromelalgia

S. Weir Mitchell first described this rare disorder, paroxysmal attacks of pain and redness of the skin of hands and feet. The pain usually begins in the feet, in the ball of the big toe and is followed by redness, throbbing, burning, and edema. The attack is aggravated by heat and dependent posture of the feet; it is relieved by cold and elevation. The attacks last from minutes to hours, are often worse at night, cyanosis may appear at the end of the attack. The disorder is more frequent in males and like Raynaud's disease is seen at times with other organic neurologic diseases. It is a true angioneurosis and may be due to a disease of the sympathetic lateral horns. In many respects it suggests an atypical type of Raynaud's disease.

Causalgia

A minor injury to a peripheral nerve may lead to chronic irritation of the sensory nerve, which causes a burning type of pain. This condition is frequently seen in war and industrial wounds.

The distribution of the pain is not always anatomic. The same symptoms may spread up a limb or to the opposite limb outside the distribution of the sensory nerve involved, without adequate anatomic explanation. Trophic changes, glossy skin, coldness, cyanosis, and edema are present.

The subjective complaints are excessive with local hyperesthesia. The motor disability may seem to be functional, or exaggerated or even malingering type. If combined with genuine functional symptoms, it may lead to an erroneous diagnosis of compensation neurosis. A severe psychiatric disorder may accompany major causalgia.

De Takats has recently described three stages in the development of the syndrome. First stage characterized by burning pain made worse by jarring, emotional upsets and air currents. The skin is warm and dry, subcutaneous tissues are edematous due to increased capillary circulation. Second stage, the pain spreads, is neuralgic in character, the affected parts are cooler, periarticular edema, and joint stiffness occur. Third stage, intractable pain spreads to other parts not anatomically distributed and atrophy of skin, muscles and bones occur. The patient becomes markedly psychoneurotic. The condition seems to be caused by chronic increased vasodilatation.

The traumatic factor is often mild, merely an injury to wrist or ankle. Usually physiotherapy does not relieve and may increase suffering. Heat, massage, and active motion aggravate the symptoms. Repeated procaine or novocaine injections into trigger points or sensitive areas gives relief in the early stages. Injections of appropriate sympathetic ganglia may also give relief but sympathectomy may be required in severe cases.

The median and sciatic nerves are those most commonly involved. In one of my patients a severe median nerve causalgia developed from injection of hypertonic solution into the antecubital vein. Fluid escaping outside the vein produced a perivascular induration and resulted in a severe, disabling median neuritis and causalgia. The condition persisted for about two years before recovery. In another case, after an accidental gunshot injury near the brachial plexus, such an intolerable causalgia syndrome of the median and ulnar nerves developed that the patient tried to persuade a surgeon to amputate the arm. Upon refusal he committed suicide.

Another patient had severe pain after a mild trauma of the foot. Surgical resection of the area only made matters worse. Later a posterior root resection, rhizotomy, and finally thigh amputation all failed to prevent the spread of the pain. Although there were many hysterical and compensation factors in the case it seemed unlikely that this patient would permit all these procedures for relief if her suffering had not been great. Complete permanent disability resulted from this major causalgia.

Phantom Limb

The illusion of persistent presence of a limb after amputation is called phantom limb. For years after the stump is healed there is a vivid sense of the absent member. The patient is often more aware of it than of the normal limb. Sometimes the pain is so distressing as to destroy his social usefulness. In a high percentage there is the sensation of contracture, nails cutting into the flesh or being pulled out, boring pain in the bones, tearing, pressing, stabbing, or burning. A slight injury to the stump may aggravate and

nervous tension, worry, fatigue states, exposure to cold, or change of weather may increase the sensations. The stump is usually colder than normal. Excess sweating and hyperesthesia may be present; always the patient perspires more on the affected side. Fibrillary twitchings and at times involuntary jerking movements occur.

Amputation of neuromata usually fails to give relief; but blocking of the sympathetic nerve impulses by repeated injections of the stellate and second thoracic nerves may give relief.

Moersch and Bailey believe the pain is central or psychic in origin and the condition is allied to obsessional neurosis. According to Livingston, the evidence favors some form of peripheral stimulation which brings about an abnormal state of the blood vessels and sweat glands with cutaneous hyperesthesia. Relief from injection of the sympathetic nerves reinforces this theory.

One of my patients after leg amputation for Buergers disease had a very distressing sensation of phantom limb, associated with a reactive depression and complete invalidism. She refused all attempts to equip her with an artificial limb. The patient made a complete recovery from the depression following electroshock therapy and ceased to complain of the phantom limb. She has remained well and active for two years.

Scleroderma

A disease of the skin characterized by diffuse or patchy induration and atrophy is scleroderma. It may occur at any age but usually begins insidiously in adolescence, more frequently in women than in men. Dermatomyositis may pass over into scleroderma. Its etiology is unknown, a "trophoneurosis." The pathology consists of atrophy and induration of the skin, subcutaneous tissue, muscles, bones, arteries, and nerves, with reduction in the capillary bed and loss or excess of pigmentation.

The disease affects every part of the body and limbs, face, neck, shoulders, arms and fingers. At first it may be circumscribed with areas of erythema, syncope, and cyanosis, as in Raynaud's disease. There is a gradual thinning of skin with atrophy, pale, marble-like tight skin, thin lips, narrow nose and pinched face devoid of expression. Anhidrosis, excessive or reduced pigmentation, itching, numbness, and loss of sebaceous glands appear. Sclerodactylia with rigid joints and trophic changes in the hair and nails occurs. The disease runs a slow chronic course with eventual cachexia.

This condition, sometimes confused with deforming arthritis offers an entirely different clinical picture; the blood studies show a normal condition and the sedimentation rate is normal, with no evidence of infection. The extremities affected appear infantile and under developed, quite different from their appearance in arthritis.

Facial hemiatrophy and progressive lipodystrophy, also rare trophoneuroses which begin in early life with progressive thinning of subcutaneous fat, are related to scleroderma. Little is known of their etiology.

Treatment

Sympathetic nerves maintain a constant tonic influence upon peripheral blood vessels and are capable of causing sustained vasospasm and probably causalgia pain, if we accept the theory of local ischemia. A release of spasm is accomplished by producing vasodilation from sympathectomy. Probably other factors in the production of these syndromes are not well understood. In causalgia chronic vasodilatation is present. Novocaine injections of the trigger zones and sympathetic ganglia, followed where relief is obtained by injection with sympathetic ganglionectomy, is the best treatment. Thoracic and lumbar sympathectomy or ganglionectomy have through permanent vasodilatation produced favorable results in Raynaud's disease, and also in some cases of phantom limb, causalgia, and intractable erythromelalgia. One per cent procaine or novocaine injections are carried out in the injured part daily. Where the pain has spread to other parts then paravertebral injections are necessary. If the pain returns within a few hours after injection sympathectomy should be performed, usually the second and third thoracic for upper extremities and the second and third lumbar sympathectomy for lower extremities.

Physiotherapy in my experience has failed to relieve the pain of these afflictions. Temporary relief has been obtained in Raynaud's disease and scleroderma from artificial fever, probably from its vasodilatory effect. Medical

therapy likewise has been of little help although in one case of Raynaud's disease and acroparesthesia I felt that nicotinic acid was of definite benefit, perhaps again from vasodilation. Estrogenic substances are recommended in acroparesthesia largely because the syndrome is seen during the female menopause.

In causalgia of traumatic origin with evidence of scar tissue, a neurolysis should be done early and a nerve transplant to a new bed. Removal of foreign bodies and scar tissue have helped in some cases where a definite trigger zone or sensitive area could be felt. In other instances in which there was every reason to believe that injury to soft tissue and adhesions were the cause of pain, surgical removal did not benefit the patient. I know of no sure way to determine which case will be benefited, but the discomfort is usually so great that one is justified in recommending exploration. Posterior root section and rhizotomy have not helped cases of severe causalgia, in my experience. Some cases are benefited by time alone.

The many problems of understanding these syndromes will remain unsolved until the neurophysiologist can give us more adequate concepts of the physiology of the sympathetic nervous system.

Clinical Observations With Electroshock Therapy*

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In presenting the following observations based on our experience with electroshock therapy, it is our purpose to stress the clinical aspects. We hope that this report may be of value to those physicians not themselves using the treatment as well as to those actively engaged in its use. We are making no attempt to present a full evaluation of our statistical results and are trying to avoid controversial speculations.

From September 1, 1941, to October 1, 1943, a series of a hundred and thirty patients, ranging in age from 18 to 58, were treated with electroshock therapy. Each patient received a preshock survey, including physical examination, x-rays of the chest and spine and routine urine and blood studies. Other special examinations were done only as indicated. Early in our experience persistent, severe depressions were considered the primary indication for electroshock therapy. Later, we added certain patients designated as "behavior problems." By these, we mean those individuals showing extreme suicidal and/or homicidal drive, with marked restlessness, agitation and tension. Frequently these patients had become feeding problems.

In agreement with other workers, the greatest percentage of recoveries were obtained in those patients primarily showing depression with retardation or agitation (manic depressive depressions and involutional melancholia). In a total of 36 manic depressive depressions, 20 "recovered," 13 "improved" and 3 were "unimproved." In 9 cases of involutional melancholia, 4 were recorded as "recovered," 3 as "improved" and 2 as "unimproved." In the patients who "recovered," a relatively small number of treatments (4 to 6) were required. Objective and subjective improvement was noted most frequently following the third treatment.

Because a number of fundamentally schizophrenic patients also showed prominent depressive features, 56 in this classification were also treated. Of these, 20 "recovered," 15 "improved" and 21 are "unimproved." An average number of 8 treatments were required in those who recovered. Improvement was noted most frequently after the third or fourth treatment. This group showed a much greater tendency to relapse than did the group showing depression without schizophrenic coloring. (Three of 36 manic depressive depressions relapsed as compared with 21 of 56 schizophrenic depressions.) Resumption of treatment while producing temporary remissions resulted usually in no lasting improvement. (In only 2 of 15 patients so treated was the improvement sustained.) It can be inferred therefore that relapse suggests a poor prognosis. It is interesting to note that in several patients show-

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ing what was called manic depressive depression, treatment resulted in the lifting of the depression and the emergence of unsuspected, latent, schizophrenic features. In these cases, it seems probable that the initial diagnosis was incorrect.

A similar group called "undiagnosed psychoses with schizophrenic and depressive features" was treated. The affective features were of such prominence that we were reluctant to place these patients in the schizophrenic group. Of 15 such patients, 5 "recovered," 1 "improved" and 9 were "unimproved." All patients in this group who received additional treatment following relapse showed no lasting improvement. These results suggest a relationship between this group and the group diagnosed schizophrenia with depressing features.

In those patients previously referred to as "behavior problems," shock therapy was instituted when ordinary psychiatric management had failed. The greater number of these were diagnosed schizophrenia or undiagnosed psychosis. These patients constituted a serious hazard to themselves as well as to others. It was impossible to provide adequate nursing care because of the acutely disturbed state. Our hope primarily was to modify the behavior of these patients in order to make them more amenable to hospital routine. We frankly did not expect recovery. However, in a surprising number of patients, the behavior was not only mollified but improvement continued to recovery. Of 28 such patients, 12 "recovered," 7 "improved" and 9 showed "no improvement." It is noteworthy that in all patients who were feeding problems, treatment resulted in resumption of voluntary food intake and marked improvement of the general nutritional state. Treatment was not successful in patients with prominent paranoid content, particularly in those with homosexual projections. On the whole, we found shock therapy an invaluable aid in the management of severe behavior problems.

During the series of treatments, a number of interesting developments occurred. These are being listed in their approximate frequency:

1) Temporary impairment of memory, especially for the details of the present illness.

2) Headache, backache and muscle fatigue immediately following treatment. (All patients with backache had re-check x-rays of the spine with negative findings in all cases.)

3) Dislocation of the jaw in 11 of 55 patients. (Early in the series, the number of dislocations was not recorded.)

4) Urinary incontinence (no bowel incontinence occurred).

5) Occasional erections.

6) Two patients had subconjunctival hemorrhages.

7) Two patients had ruptured superficial blood vessels on the temples immediately under the electrodes.

8) One patient who by mistake had eaten breakfast vomited during the convulsion without ensuing complications. Nausea was an occasional complaint.

9) One patient complained of radicular pains in the arms (re-check x-rays of the spine revealed no fracture and the pains disappeared with physiotherapy).

10) One death occurred in the series but its relation to electroshock therapy is debatable. This patient received a series of 8 electrically induced convulsions. For some unknown reason, no further convulsions could be induced. Eleven days after the last electroshock treatment, he was given metrazol. Thirty-six hours after the metrazol convulsion—while in a cold pack given to control his excitement—the patient suddenly went into shock and died. Permission for autopsy was not obtained and the death was attributed to exhaustion incident to schizophrenic excitement.

The development of a definite hypomanic reaction occurred in only two depressions following treatment. One was of short duration while the other was persistent. Feelings of well being and relief were frequently observed but were not sufficiently marked to be considered pathological.

Three patients with marked neurotic excoriations were treated. The skin lesions had failed to respond to persistent local treatment. However, dramatic improvement of the skin lesions occurred along with improvement in the mental state.

In 17 patients it was considered advisable to administer curare* intravenously in conjunction with treatment. Our indications for its

*Intocostrin (Squibb), extract of unauthenticated curare, physiologically standardized so that each cc. had a potency equivalent to 20 mg. of standard drug, was used.

use were:

1) Age (over 45-50).

2) X-ray evidence of bone pathology (osteoporosis, old fractures, old osteo-myelitis).

3) Convulsive muscular contractions of such severity that injury was feared.

The results with curare were uniformly satisfactory and its use was without complication, except for a slight increase in cyanosis and post-convulsive apnoea. In one patient, severe nausea and retching occurred but not until after the third injection of the drug. Our dosage varied from 2½ to 3½ cc. (50 to 70 milligrams) depending on the weight and individual reaction of the patient to curare.

Summary

1) Clinical observations made during the treatment of 130 patients treated with electroshock therapy are reported.

2) Indications for electroshock therapy are presented.

3) Comparison is made of its value in the treatment of depressions in both the affective (manic depressive depressions and involutional melancholia) and schizophrenic reactions.

4) Shock therapy has proved to be an invaluable aid in the management of certain "behavior problems."

5) Complications encountered during treatment are outlined.

6) The use of curare is discussed.

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CHARLES D. ARING, M.D., *Editor*

Presentation

The patient was a 17¾ year old white male. Born prematurely, he weighed five pounds five ounces at birth. Pregnancy had been normal except for hives which occurred just before and during the early months of gestation. Copious administration of adrenalin resulted in symptomatic relief. Patient had suffered a "blue spell" on the third day of life, and until the age of 13 years he exhibited frequent periods of what was called "breath-holding," during which he would become cyanotic and lose consciousness. It is estimated that he had between 125 and 150 of these spells, occurring less frequently in later years, with none after the age of 12. Occasionally they occurred during sleep. It was thought that the attacks were precipitated by crossing the child, and he was known to have them on passing a cemetery, which he did not like to enter. Abandoned laughter precipitated attacks. In these episodes his head would be thrown backward and he lapsed into unconsciousness. The extremities would extend and the hands pronate. He was not incontinent. Any attack would last less than a minute and was followed by deep sleep. He never had a clonic convulsion. Because of the episodes of "breath-holding" the thymus gland was examined in infancy by roentgen ray and was found to be enlarged. He was given an occasional thymic irradiation, the last at about the age of 5 or 6 years. He had received a total of 20 such treatments.

As an infant he was a difficult feeding problem. He weighed 15 pounds at one year of age. Despite a voracious juvenile appetite he never gained weight normally. Because of frequent attacks of what was termed "bronchitis" or "asthma," he was skin-tested for all of the common foods at the age of 7, and was found to react positively to practically all of them. In spite of these reactions he was allowed to eat mostly as he wanted.

Beginning at the age of 5 months there occurred the feverish attacks, the final one of which was associated with his demise. Over the years there had occurred 15 to 20 of these episodes during which he had what seemed to be bronchitis, and during which a diagnosis of pneumonia usually was made. The attack was ushered in usually by what appeared to be rhinitis, and a low grade fever

which after a few hours would suddenly rise to 105 or 106 degrees F. Occasionally a chill preceded the fever. Rales could then be heard in the chest. The fever would range between 101 and 106 degrees for 24 hours and then drop precipitously, but the temperature would not return to normal. Rapid swings of fever between a range of 2 degrees would then occur for the next three or four days, the temperature tending toward normal with the passage of time. It was unusual to find the temperature the same on any two successive readings taken as closely as ½ hour apart. The later fever attacks were quite definitely aborted by ¾ grain of nembutal taken once or twice.

All attacks were accompanied by what had been called pylorospasm. He would have severe abdominal pain and pain in both arms and legs. The pain was accompanied by vomiting, and he then was unable to keep anything down for a day or two. The longest episode of vomiting lasted for ten days. The vomiting of blood during these episodes had at one time resulted in a diagnosis of gastric ulcer. He would have a normal blood count during the feverish episodes, during which the urinary output decreased, and the urine contained albumin, and hyaline and granular casts; the blood non-protein nitrogen would rise (as high as 88 mg. per cent). The blood pressure, always labile, would not rise, and usually fell during an attack. He lost 8 to 10 pounds with each attack, which he would regain quickly. The attacks were terminated by his unexpected announcement that he felt well.

His family which included an intelligent and observant physician noted that the attack of fever always followed excitement or tension. Contemplation of an impending examination in school was probably the most frequent offender, although they had occurred after the excitement of attending a football game, or precedent to a trip or before coming home after a period away from it.

Incidental observations included the fact that the eyes never teared during crying. He suffered from enuresis up to the age of 4. He was always considered to have a degree of muscular incoordination. He was unable to swim, could not run though he played at baseball; and he was adept at the piano. In his final summer it was observed that he staggered for some minutes after alighting from a subway or on coming out of darkness into the light. This was particularly disabling; it appeared as though he could not adjust readily to any rapid change and coming from darkness into light was one of them. Going from the light into the dark did not seem to affect him adversely. Loud noises frightened him, and he was always considered to suffer from "vasomotor instability".

Course: Eleven months before death when nearly 17 years old, he was 4′ 11″ tall and weighed 76 pounds, and had the appearance of a 10 or 11 year old boy. The abnormal neurological signs were the following: The retinal arteries were tortuous. The eyes did not converge. There was weakness about the mouth; when asked to show the teeth the upper lip was drawn laterally, the lower moving little if at all. The speech was of the bulbar type, guttural in sound as though the mouth were full. There was some drooling of saliva. He could not whistle. He was left handed, while his family was right handed. Muscular development was retarded and all movements lacked vigor. Breathing was largely diaphragmatic. There was lordosis of the lumbar spine and scoliosis of the dorsal spine. Resistance to passive movements was generally reduced. The range of passive movement of the joints was limited. He could not raise the hands over the head. He walked stiffly with the legs apart, weaving occasionally to either side, the trunk bowed forward somewhat. It was impossible to obtain any of the deep reflexes.

He attended high school and took part in modified physical activities. He was bedfast only during the feverish episodes, the last of which had its onset ten days before death. He had taken his mid-semester examinations. There then began the usual rhinitis and on the eighth day the usual vomiting and abdominal cramping. The vomiting became retching and the temperature ascended sharply to 103° F. As always during these attacks he appeared desperately ill. Nembutal which had ameliorated some of the previous feverish attacks failed to be effective. His fever rose to 107° F. About 12 hours before death he was given morphine sulphate, grain ⅛, after which he could never be aroused. This was the first dose of morphine that he had ever had. He ceased to excrete urine. Rales ap-

peared in the chest and he expired.

Laboratory Data: Psychological examinations revealed intelligence quotients of 99 at the age of 5 years, 92 at 6, and 88 at the age of 13 years. Blood counts were normal. Blood Wassermann and Kahn tests were negative. At the age of 6 the sugar and non-protein nitrogen content of the blood were normal. Numerous roentgen studies of the chest and gastroenteric tract showed nothing abnormal.

During a feverish episode at the age of 17, the NPN done repeatedly rose as high as 49 mg. per cent, and on another occasion to 88 mg. per cent. For many years the urine contained casts which disappeared at the age of 13, to recur in the feverish episodes. Albumin was sporadically found in the hundreds of samples that were examined throughout his life. The highest concentration in these routine samples was 1.025, the lowest 1.010.

The blood pressure varied very widely. Determinations during a day at the age of 9 years were before breakfast 88/40; at 3 P. M. 180/130 in his father's office; at 6 P. M. 128/60 at home; at 7 P. M. 150/100 at home, and at 8:30 A. M. on the following day 150/114. This variance continued throughout life. On one occasion at the age of 16 it was 185/150; two months later 128/80 and on the same afternoon at a ball game 152/100. The highest measurement was 220/160 less than one year before death. A day later the blood pressure was 120/80.

Differential Diagnosis

Dr. George Engel: This is a very difficult case and I spent a lot of time wrestling with it. I secured more data from Doctor Aring and from other hospital records, which I felt were essential for the interpretation of the case. On looking closely into the record of the case it becomes apparent that we are dealing with a non-progressive, episodic disease with its onset in infancy. The nature of the attacks is discrete and apparently each attack resembled the preceding one. It is stated that all attacks were provoked by emotional excitement. Each attack began abruptly, followed by elevation of temperature for several days with marked fluctuations in temperature during this period, and finally defervescence over the course of two or three days. Additional data revealed that with the onset of these attacks the patient would complain of coldness, he would shiver, and sometimes he had a chill. There was associated pallor of the face and extremities. The hands and feet were cold, the trunk warm. Sweating was absent. Piloerection was not noted. Data from the nursing notes of one hospital admission revealed that the sudden defervescence in temperature was marked by sudden profuse sweating, warm extremities, and flushing. The attacks were also associated with vomiting, nausea, diarrhea, and pain in the extremities. There were formed elements, casts, and albumin in the urine during the attacks, and the NPN generally rose. Blood pressure fluctuated between hypertension and hypotension, but there was no consistent trend during the attack. Attacks were said to be aborted by nembutal, but it was also reported that the fever was brought down with antipyretic drugs. I also learned that this child had a high temperature during measles and chickenpox. In all these attacks the child was described as being very ill and during the period of elevated temperature he sometimes looked almost moribund.

Since the attacks of fever are important in the case, I shall digress to talk briefly about the mechanism of heat regulation. It is a remarkable thing to me that although all physicians are aware that the hypothalamus has something to do with heat regulation they seem to regard it as a sort of furnace that is periodically turned on or off. In all the clinical literature that I have found concerning temperature disturbances due to hypothalamic lesions, there is little data concerning the status of the peripheral mechanisms of heat dissipation or conservation. Actually it is quite obvious that heat regulation is mediated chiefly by peripheral mechanisms. Body temperature depends upon a balance between heat dissipation and heat conservation and formation. Heat dissipation is mediated through the mechanisms of radiation, convection, and conduction, and evaporation of water from the surface. Any body will radiate heat to neighboring structures which are at a lower temperature. Physiologically this is controlled by vasodilatation. By increasing the blood flow to the skin a greater volume of circulating blood is brought nearer to surface and hence there is greater turn-over in heat dissipation. Evaporation of water from the surface leads to cooling. This

is mediated chiefly through the mechanisms of sweating. A small amount of evaporation from skin takes place without sweating, the so-called insensible water loss. Those are the specific mechanisms of lowering body temperature. Heat conservation is effected through (1) piloerection, which is not an effective means of heat conservation in humans, and (2) peripheral vasoconstriction. In addition the body can raise its temperature by increased heat formation. The most common mechanism is by increased muscle activity or shivering, which may become so marked as to constitute a chill. Also there may be an increase in general cellular metabolism. The latter plays an important role in the fever of infections, reaction to foreign proteins, etc. Each of these factors may be modified in a number of ways. When environmental temperature exceeds body temperature, radiation, convection, and conduction can no longer be effective and evaporation, through sweating, plays the major part. Peripheral factors which alter circulation and sweating will affect body temperature. Any morbid process or drug which causes peripheral vasoconstriction may cause the body temperature to rise. Similarly, drugs that increase sweating or decrease peripheral circulation may lower body temperature. It is a common phenomenon to see terminal rise in body temperature to 104-105° just before death. These patients generally have marked pallor, vasoconstriction, cold hands and feet, and actually are in shock. But their ability to dissipate heat is impaired because of the intense vasoconstriction, and in spite of cold skin, rectal temperature may be high.

In studies of the central regulation of temperature it has been well established that heat regulating centers are located in the hypothalamus. In the anterior portion are located nuclei that have to do with heat dissipation. Stimulation of this area results in sweating and vasodilatation. The destruction of this region results in hyperthermia, due to relative overactivity of the centers of heat conservation and formation. The latter functions are located in the posterior portion of the hypothalamus, stimulation of which results in piloerection, shivering, and vasoconstriction; destruction resulting in hypothermia. When both these hypothalamic centers are destroyed, poikilothermia results.

From the clinical data on this patient, it is apparent that all means of temperature regulation were intact, but there seemed to have been some imbalance. The problem is to consider what might provoke this disturbance in heat regulation.

First it is necessary to rule out the possibility that there was an infection of some sort. I am unable to think of any infection that would start at the age of 5 months and last 18 years, with periods of relative well-being between the attacks. It does not resemble malaria or any low grade chronic infection, such as brucellosis. Further, the temperature course is very unusual for any infection. In fever due to infection, once the initial chill is past, the patient generally is flushed, sweating, and feels hot. During the periods of elevated temperature, this boy was described as being cold, pale, with cold hands and feet, and not sweating. Although this may appear a very inadequate consideration of infection—the most common cause of fever, the atypical course makes me quite confident that an explanation will not be forthcoming on that basis.

Next, one might consider fever due to some defects in the peripheral mechanisms of heat dissipation. This brings up a condition known as congenital ectodermal dysplasia with anhidrosis, a rare condition in which the absence of sweating results in high fever when the environmental temperature is high. This child did sweat, so that is not an important consideration. The next possibility is whether these paroxysms of fever may have been related to extreme peripheral vasoconstriction due to humoral factors, particularly epinephrine. Pheochromocytoma of the adrenal medulla characteristically provokes paroxysms of extreme vasoconstriction. The usual symptoms described are paroxysmal hypertension, palpation, flushing of face, followed by blanching, nausea, vomiting, and pounding headache. In most instances the attacks are of short duration. With attacks lasting several hours, however, the prolonged and extreme peripheral vasoconstriction leads to marked disturbances in body temperature. I once had the opportunity to study temperature changes in a 23 year old girl with a pheochromocytoma whose paroxysms lasted as long as 48 hours.[1] Initially the symptoms were as I have just described, but after a few

hours the temperature of the hands and feet would fall from 90° F. to 76° F. (32° C. to 25° C.), whereas her rectal temperature would rise from 98° F. to 105° F. (37° C. to 40° C.) by the end of the first day. The rectal temperature would remain up as long as the extreme vasoconstriction persisted. She sweated profusely from the trunk but in spite of this, the peripheral vasoconstriction was so marked that this girl was still unable to dissipate heat adequately. Under these circumstances her rectal or internal temperature rose. This disease might seem like a good possibility in the boy under discussion. However, in the case of pheochromocytoma the rise in temperature is secondary to rise in blood pressure and to diminished peripheral circulation. In this boy there is no record that the blood pressure rose before the temperature. Indeed there was no consistent association between blood pressure and temperature. In addition this boy lacked certain of the primary symptoms resulting from epinephrine. He had no headache, no pupillary changes, no tachycardia, and no sweating during the period of vasoconstriction. The course of the individual attacks and the course of the disease were not that of pheochromocytoma.

The next consideration is whether or not these attacks could be provoked by any central disturbance in temperature regulation. If one proceeds up the neuraxis, one is unable to find any area caudad to the hypothalamus, in which structures and tracts are closely enough grouped to yield generalized disturbance in heat regulation, without involving other structures in the central nervous system. One finally gets up to the hypothalamus and one is immediately struck by the similarity between these attacks, and those described as the result of stimulation of the posterior hypothalamus or destruction of the anterior hypothalamus. In addition, the fact that these attacks are prevented by nembutal focuses attention on the hypothalamus. It is a fact that nembutal will inhibit the heat formation and conservation centers in the posterior hypothalamus. However, we also know that this child had flushing and sweating. This suggests that at first the posterior hypothalamus was stimulated with resultant shivering, vasoconstriction and hyperthermia, and that then the anterior hypothalamus was stimulated with sweating, flushing, and fall in temperature. There must therefore have been some disturbance in the relationship between the two portions of the hypothalamus concerned with heat regulation.

At this point the question might be raised as to whether this child had any other signs that might be ascribed to hypothalamic disturbance. Gastrointestinal symptoms such as described in this patient during his attacks have been demonstrated in animals on stimulation of the anterior hypothalamus. Fluctuating hypertension suggests involvement of blood pressure regulatory mechanism in the posterior hypothalamus. Less certain to be of hypothalamic origin are the attacks of breath-holding followed by somnolence, which in certain respects resemble narcolepsy or cataplexy. Hyperphagia has been produced in animals by hypothalamic lesions, but these animals usually become very obese. This patient was almost a dwarf. So far I am not aware of the combination of hyperphagia and dwarfism being produced by hypothalamic lesions. Thus there are a number of symptoms which may definitely be related to the hypothalamus and others which might be.

To summarize so far, we are dealing with a non-progressive, episodic disease in which our data suggest the possibility of some disturbance in hypothalamic function. Before proceeding further, we might pause and consider the possible etiological lesions in the hypothalamus.

In the first place it seems quite clear that one cannot postulate any fixed lesion in the hypothalamus, as we already have demonstrated that both parts of the hypothalamus appear to be capable of function. That would rule out infarction, gliosis, or degenerative lesions destroying part of the hypothalamus. Neither can the disorder be explained as an expanding lesion. The hypothalamus is located in a crucial area of the brain. An expanding lesion could hardly last for 18 years, without leading to increased intracranial pressure or hydrocephalus, or to involvement of the optic chiasm, or to diabetes insipidus, or the adiposo-genital syndrome, or pubertus praecox, or involvement of the adjacent internal capsule. None of these things was present and there was no progression so that it appears to me that neoplasm is ruled out. There is also nothing in the history to suggest an inflammatory lesion such as that due

to encephalitis at a very early age, which again brings back a fixed lesion in the hypothalamus, further there is no history to suggest this. One might wonder about a vascular lesion, which permitted a certain degree of variation in blood flow to this region from time to time. An aneurysm without neighboring signs and without subarachnoid bleeding would be unlikely. One might consider a vascular anomaly, such as a cavernous hemangioma or some type of congenital arteriovenous shunt, but the more I thought about it the less able was I to visualize just how such a lesion could produce this picture. Pineal or pituitary tumor I have already eliminated from consideration.

Now that I have ruled out all types of lesions of the hypothalamus, I am not left with very much. I am faced with the conclusion that the hypothalamus is impaired through a process not located in the hypothalamus. Could there be a lesion higher up in the brain involving connections to the hypothalamus? My neuroanatomy fails me at this point, and I am unable to name any region about the hypothalamus, destruction of which would lead to such a picture. Nevertheless I think we can say that there is some functional derangement of the hypothalamus. I think this is a consideration worthy of further thought. We have already presented considerable evidence to make us feel that the disturbance is central in origin. We know also that these attacks are described as being provoked by emotional factors. We know also that there was an abnormal temperature reaction to the exanthems. If we are dealing with some type of functional disturbance involving hypothalamus the question comes up as to whether this is the primary disorder or whether it is secondary to some other process. What evidence is there for any other process not related to the hypothalamus? I believe this boy had a number of signs and symptoms which may have been of different origin. For one thing he had a generalized disturbance in his muscles. The muscles were small and somewhat weak and there were absent deep reflexes, lordosis, scoliosis, bulbar speech without evidence of bulbar palsy, drooling, and defective convergence. In the absence of any evidence of lesion of the spinal tracts or polyneuritis, I am forced to conclude that the difficulty was located in the muscles themselves, perhaps some type of muscle dystrophy. Dermatomyositis might be considered, but there is nothing in the history to favor this. Metabolic disturbance with derangements in creatine or creatine metabolism could also be thought of. One might wonder about an endocrine disturbance. It is known that the male sex hormone has to do with muscular development and the patient had an undescended testicle. Whether the pituitary had anything to do with this. I cannot say. I doubt it. I think the question of the muscular weakness may be related to the dwarfism. Essentially the boy was a symmetrical dwarf. This was not confined to the muscles because the skeleton was also small. In the absence of x-rays we know nothing of such important matters as bone age, epiphyseal closure, etc. Further he had absent secondary sex characteristics, no pubic hair and undevelopment of the external genitalia. Could this be related to some disturbance in the pituitary? I believe this may more readily be explained by the extreme weight loss resulting from the repeated episodes of fever. He was said to lose from 8 to 10 pounds with each attack. With such a handicap it is obvious that this boy had to gain more than a hundred pounds extra just to make up his periodic weight loss. I believe this is adequate explanation of the underdevelopment and also of his hyperphagia. It may also account for the muscular dystrophy. In short, these manifestations most likely were indirect results of the central disturbance.

Then we have the factor of allergy and asthmatic attacks. I think these are incidental and are not essentially related to the picture as a whole. The family allergic history was strong.

The description of arterial changes in the fundi, periodic hypertension, and evidences of impaired renal function during the attacks makes me wonder if he had some type of primary renal disease. Between attacks, his kidney function was adequate. I am inclined to believe that the vascular changes were secondary to hypertension, which was central in origin.

In final analysis then, we find a condition in which most, if not all, the findings might be caused by some disturbance in the hypothalamic function, yet I am unable to ascribe it to a lesion in the hypothalamus, while the

neuroanatomic data available to me do not enable me to locate a lesion about the hypothalamus. That brings up an interpretation which I am sure is going to get me into trouble. I am going to be so bold as to suggest that these attacks were psychogenic in origin and that the tissue changes were secondary to such a mechanism. The protocal repeatedly emphasizes the role of emotional factors in provoking the attacks. We know perfectly well that psychogenesis can lead to organic lesions as the result of longstanding disturbance in function. Ulcerative colitis, peptic ulcer, hypertension are but a few examples. Is there any evidence that paroxysms of fever resembling those seen in this case may be provoked by psychological factors?

This is a domain of psychosomatic medicine that has received relatively little attention. I can cite from my own experience three cases characterized by repeated episodes of chilling, fever, gastrointestinal symptoms, oliguria, and limb pains over many years, and the most exhaustive study failed to reveal any infectious cause but psychological precipitating factors were prominent. These cases were all in adults and as far as I know all are still alive.

There is in the literature the report of a 43 year old man who had frequent recurrent bouts of fever up to 104° F. for 13 years, whose attacks ceased after psychotherapy.[2] Alt and Barker[3] described a case of intermittent fever that began at the age of four years and had lasted 25 years at the time the report was made. And finally Dunbar[4] cites considerable foreign literature on psychogenic fever, including instances in which marked temperature changes were induced by hypnosis.

With these data, plus the previous line of reasoning which has led us to conclude that the disturbances in temperature regulation in this case are due to a defect in hypothalamic function without a lesion in the hypothalamus, I am going to suggest that the attacks were of psychogenic origin and that the anatomical changes that Dr. Scheinker will demonstrate may be secondary to longstanding derangement of function. The hypothalamus will be intact. There may be a lesion (vascular or degenerative) near the hypothalamus, but where I do not know. There may be vascular lesions secondary to the longstanding intermittent hypertension, including changes in the aorta and early small vessel lesions, perhaps in the kidney, heart, and perhaps in the brain. The muscles should show some atrophic or degenerative changes. There may be acute changes in the gastrointestinal tract, particularly erosions. There will be changes in the lungs related to the longstanding asthma, and perhaps more acute changes of the terminal illness.

Clinical Diagnosis

Hypothalamic attacks of undetermined etiology.
Amyotonia congenita.

Doctor Engel's Diagnosis

Hypothalamic attacks of psychogenic origin.
Unlocalizable lesion (vascular or degenerative) near the hypothalamus.
Erosion of gastroenteric tract.
Asthmatic bronchitis and terminal lobular pneumonia.

Anatomical Diagnosis

Destruction of the inferior half of the dorsomedial nucleus of the right thalamus by an old cyst of softening and gliosis.
Amyotonia congenita.
Acute necrosis of the liver.
Status thymolymphaticus.
Asthmatic bronchitis and minimal acute lobular pneumonia.
Minimal glomerulonephritis.

Discussion of Pathology

Dr. I. Mark Scheinker: I shall start with the visceral changes and then consider the alterations of the central nervous system. There were changes that may be called status thymolymphaticus. Except for a mild congestion, no pathologic findings could be disclosed in the adrenal glands. In the lungs the outstanding change was marked thickening of the bronchial walls which in part was due to proliferation of connective tissue, and in part a dense accumulation of inflammatory cells. These cells appeared at first glance to belong chiefly to the lymphoid series, but on closer inspection many were seen to be eosinophils. Beneath the intact bronchial mucosa there was an abnormally thick, hyaline, basement membrane. The air sacs were generally free of exudate; some alveoli con-

tained leukocytes only in the immediate vicinity of bronchial radicles. We are dealing with an allergic form of bronchitis and associated minimal acute lobular pneumonia. The alterations found in the kidneys may be classified as low grade glomerulonephritis, with occasional scarring of some of the glomeruli. There were focal areas of acute necrosis in the liver. I was inclined to think that these areas of necrosis occurred within the last few days of life and might have been, in part at least, the result of the single dose of morphine, since he had never had this drug previously and since it appeared to initiate the coma from which he never revived.

In the central nervous system, the most notable feature was a small cystic cavity in the right thalamus. This cavity measured 9 by 1½ by 2 mm., and was confined to the dorsomedial nucleus. Inferiorly it reached to within 3 mm. of the ependyma of the third ventricle. The nervous tissue adjacent to the cavity appeared homogeneous in structure and slightly firm to touch. The entire lower half of the dorsomedial nucleus of the right thalamus was involved. The remaining parts of the diencephalon did not disclose any gross abnormalities. The hypothalamus, brain stem, and cerebellum appeared grossly normal. The spinal cord showed changes consistent with of amyotonia congenita, the muscle did not.

The case presented here is unique as regards the small size of the lesion and its rather discrete localization. It is significant since it clearly indicates that the hypothalamus is under direct control of higher mechanisms.

There is a single case in the literature that could be compared with it, a patient described by Penfield in whom the symptomatology contained: restlessness, sudden vasodilatation of the face, rise of the blood pressure, lacrimentation, diaphoresis, salivation, dilatation or contraction of pupils, transient shivering, and Cheyne-Stokes respiration. These attacks were associated with a tumor of the third ventricle. It pressed against the anterior nucleus of the thalamus on both sides. The fornices were stretched over the tumor. There must have been pressure on the mamillothalamic fasciculus, anterior commissure, and anterior peduncle of the thalamus. The movable tumor acted as a ball-valve, shutting off the flow of cerebrospinal fluid through the foramina of Monro, and transient hydrocephalus resulted.

The case presented here represents the first clear evidence in man that a circumscribed, focal lesion confined exclusively to the dorsomedial nucleus of the thalamus may result in a hypothalamic syndrome. Analysis of the clinical symptomatology suggests that the unilateral thalamic lesion was associated in the production of paroxysmal discharge through the vegetative nervous system; comparable to the focal discharge through the cerebrospinal system, known as Jacksonian fits.

The frequent attacks of "breath-holding" or "blue spells," during which the patient would become cyanotic and lose consciousness may have been autonomic seizures chiefly involving the mechanisms for regulation of the respiratory and vascular systems. In contradistinction to the disturbances of temperature regulation associated with hypothalamic lesions, which is manifested as constant hyperthermia or hypothermia, in our case there was severe maladjustment of these mechanisms under stress.

Dr. Charles D. Aring: Psychological factors did certainly provoke many, if not all of the feverish attacks. Their relation to what were for this boy, strenuous situations, were repeatedly demonstrated to the satisfaction of everyone who had anything to do with the patient. There can hardly be any question that psychogenetic factors initiated the terrific attacks, the final of which was fatal. I should say that Doctor Engel has analyzed this case expertly, in fact better than any of us who saw him (about 100 physicians throughout the country).

Dr. Engel: I am not nearly as chastened as Doctor Scheinker evidently thinks I should be. Actually I feel very well satisfied. As Doctor Aring has emphasized all the attacks were provoked by emotional factors. Now it is well established that the hypothalamus is the main-way station for the visceral and somatic expression of emotion, although obviously the ideational content arises from higher levels. In this instance we are dealing with a release phenomenon in the sense of Hughlings Jackson. Because of the interruption of certain higher pathways by this lesion we see over-reaction of the hypothalamus to more or less normal emotional stimuli. Sim-

ilarly there was over-reaction to certain physiological stimuli, as is attested by his reaction during measles and chicken pox. I suspect that if this boy could have lived in a vacuum, he might never have had any symptoms (like Bard's hypothalamic cats which were symptomless unless stimulated). I prefer this interpretation to the one advanced by Doctor Scheinker that the lesion acted as an irritative focus and provoked "autonomic epilepsy".

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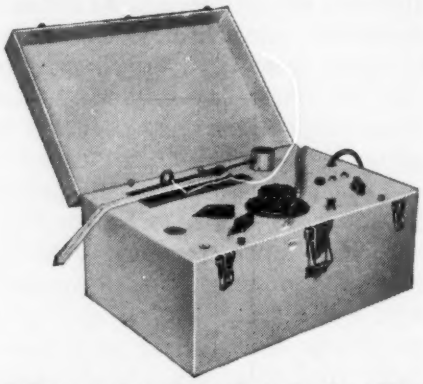

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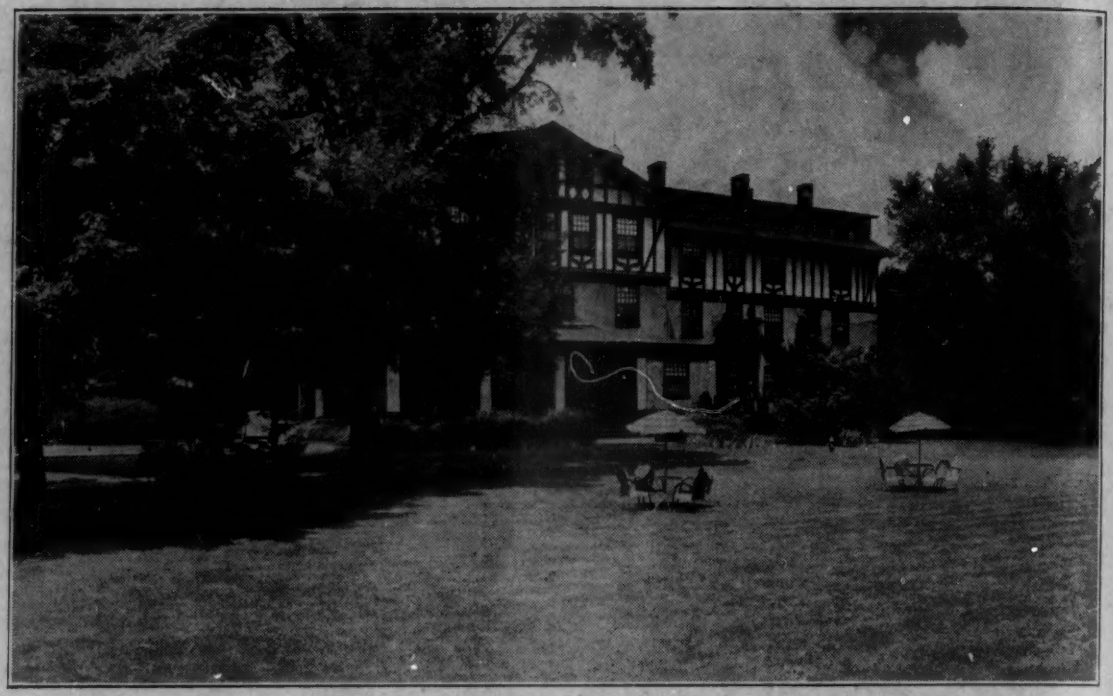

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